and *Clostridium novyi* α-toxin (Bette, P., et al., Toxicon 29 (1991) 877-887). Enterotoxin A and cytotoxin B have been characterized by Sullivan, N.M. et al., Infect. Immun. 35 (1982) 1032-1040, von Eichel-Streiber, C., et al., Microbiol. Pathogenesis 2 (1987) 307-318. Toxin A and toxin B are glucosyltransferases which modify threonine 37 of the GTPase Rho. By attracting of glucose at this position of Rho, this GTPase is blocked in its function. Recently, toxin B and toxin A from *C. difficile*, the causative agent of antibiotic-associated diarrhea (Lyerly, D.M., et al., Clin. Microbiol. Rev. 1 (1988) 1-18), were shown to covalently modify the mammalian protein Rho by UDP-Glc dependent glucosylation of threonine 37 (Just, I. et al., Nature 375 (1995) 500-503; Just, I., et al., J. Biol. Chem. 270 (1995) 13932-12936). Rho is a small ras related GTP-binding protein involved in the control of actin polymerization (Hall, A., Ann. Rev. Cell Biol. 10 (1994) 31-34). Glucosylation of threonine 37 of Rho by *C. difficile* toxins A or B apparently inactivates this protein and results in a loss of actin stress-fiber assembly.

IN THE CLAIMS

Please amend claim 21 as follows.

21. (Twice Amended) An isolated polypeptide fragment of *Clostridium sordellii* lethal